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Chronic intermittent hypoxia aggravates palmitate-induced lipid accumulation and apoptosis in a NAFLD cell model

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BACKGROUND/AIMS: Obesity is a risk factor for nonalcoholic fatty liver disease (NAFLD) and obstructive sleep apnea (OSA). Yet, the involvement of OSA in the pathogenesis of NAFLD is debatable. Cardinal signs of OSA are recurrent episodes of severe hypoxia and reoxygenation, leading to chronic intermittent hypoxia (IH). This study was aimed at examining the hypothesis that chronic IH could aggravate lipid accumulation and apoptosis induced by sodium palmitate in HepG2 cells, a model for NAFLD. MATERIALS AND METHODS: Cultured HepG2 cells (85 % confluent) in DMEM (with 10 % FBS and antibiotics) were divided into normoxic (Nx) (at a humidified atmosphere with 5 % CO2 and 95 % air) and intermittent hypoxia (IH) groups (with 5 %CO2 and alternating oxygen levels between 21 %and 1 %every 12 h). Also, cells were treated with sodium palmitate (0.5uM) or vehicle for 48 h. Cell viability was determined by colorimetric MTT assay and apoptotic cell death was measured by propidium iodide staining. Oil red O staining was used to assay the intracellular lipid deposit and DCFDA fluorescence staining was used to detect the reactive oxygen species (ROS). RESULTS: Sodium palmitate significantly decreased the cell viability and the effect was much greater in the hypoxic group than that of the normoxic group. In addition, levels of lipid deposit were increased in the palmitate-treated group and were remarkably more in the hypoxic than that of the normoxic group. Moreover, ROS levels were significantly elevated in the hypoxic group which were enhanced by the palmitate treatment. Apoptotic cells were significantly increased in the palmitateor hypoxia-treated group and were doubled in the co-treated group. CONCLUSION: Chronic intermittent hypoxia aggravates palmitate-induced lipid accumulation and apoptosis in HepG2 cells, suggesting a significant involvement of CIH-induced oxidative stress in the pathogenesis of NAFLD at the cellular level.